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Primary Hyperparathyroidism: Report of Cases in Asymptomatic Older Adults in Honduras

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Abstract

Introduction: Primary Hyperparathyroidism (PHPT) is characterized by hypercalcemia, hypophosphatemia, and elevated PTH levels. The profile is a woman over 50 years of age, postmenopausal, and is detected in her asymptomatic presentation by performing screening studies measuring calcium and serum phosphates. The 85% of cases are asymptomatic and are due to a PTH-producing parathyroid adenoma. The symptomatic case may have kidney damage, osteoporosis and cardiovascular involvement. Imaging studies are performed to look for adenomas; management can be surgical or conservative.

Clinical case one: 77-year-old female, with arterial hypertension, asymptomatic. He went for routine evaluation with vitamin D 10.6 ng/ml, serum creatinine 1.7 mg/dl, serum calcium 11.9 mg/dl, albumin 4.5 g/dl, phosphorus 2.26 mg/d, PTH 163.30 pg/ml, ultrasound neck without masses, normal abdominal ultrasound and normal electrocardiogram. Conservative management with bisphosphonate was decided.

Clinical case two: 75-year-old female, hypertensive, obese, and asymptomatic with routine tests with calcium 12 mg/dl, phosphorus 2.36 mg/dl, vitamin D 17.1 ng/ml, PTH 340 pg/mL, electrocardiogram with sinus bradycardia, Normal abdominal ultrasound, and thyroid ultrasound with a 1.4 cm by 1.6 cm nodule. Conservative management with bisphosphonate was decided.

Conclusion: Screening should measure calcium, phosphorus and PTH levels in patients over 50 years of age. In asymptomatic older adults, conservative management can be performed and in symptomatic patients, treatment is surgical.

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Introduction

Hyperparathyroidism is a common endocrinological problem; it can be primary if it is due to increased secretion of PTH (parathyroid hormone) in the parathyroid gland due to a tumor, or secondary (associated with chronic kidney disease) [1].

Primary Hyperparathyroidism (PHPT) is characterized by hypercalcemia, hypophosphatemia, and elevated PTH levels. Elevated PTH levels lead to hypercalcemia through increased tubular calcium reabsorption, osteoclast-mediated bone resorption, and increased intestinal calcium and phosphate absorption [2,3].

Epidemiology

The incidence of the disease is variable depending on the geographic region. In the United States of America, the incidence in those under 45 years of age is the same in men and women, but after 45 years it is higher in women, with the black race predominating, with a low incidence in Latinos and Asians; the incidence is 233 per 100,000 in women and 85 per 100,000 in men, increasing at 70 to 79 years to 1,409 per 100,000 in women. More are diagnosed asymptomatic due to early diagnosis through screening tests. In Europe, like the US, a greater number of asymptomatic patients are detected by periodic calcium measurement, and there are more cases in women over 50 years of age. In South America and Africa, a greater number of symptomatic patients with high levels of serum calcium have been seen compared to the US, this is due to fewer screening tests and the fact that reports are based on tertiary centers and not on population studies. As we can see, the profile is a woman over 50 years old, postmenopausal, and it is detected in her asymptomatic presentation by performing screening studies measuring calcium and serum phosphates [4].

The age at which it occurs guides us to the possible cause. In people between 25 and 30 years old, we should suspect hereditary or familial forms such as Multiple Endocrine Neoplasia type 1

(MEN1), Multiple Endocrine Neoplasia type 2A (MEN2A) and Hyperparathyroidism-Jaw Tumor (HPT-JT). These syndromes occur with a variety of tumors (endocrine and non-endocrine), including cancer [5,6].

In people over 50 years of age, 80% to 85% of cases of PHPT derive from parathyroid adenomas, single in most cases, and 15% to 20% are due to hyperplasia of the four parathyroid glands. The rarest causes are parathyroid carcinoma and parathyroid cysts, which occur in between 1% and 2%. The best-known risk factor is exposure to neck radiation during childhood [7,8].

Clinical manifestations

Currently, 85% of PHPT cases are asymptomatic, this is due to the periodic measurement of calcium and phosphorus levels in the routine control of older adults, making the presentation of severe forms of the disease less frequent. The clinical manifestations are a form of late presentation; Among the clinical forms of presentation, we have renal manifestations that include nephrolithiasis and chronic renal failure due to nephrocalcinosis [9,10].

At the bone level, it can debut with fractures due to a decrease in bone mineral density. A rare form today is osteitis fibrosa cystica, whose clinical manifestations appear as bone cystic lesions, skeletal deformities and bone pain [11].

Untreated PHPT is also related to cardiovascular disease; between 40% to 60% have concomitant arterial hypertension, left ventricular hypertrophy and calcification of heart valves. The pathophysiological mechanisms are not clear, but there is a relationship with increased cardiovascular mortality. At the digestive level it is related to nausea, anorexia, constipation, heartburn, peptic ulcer and more serious acute pancreatitis, the latter a rare form of presentation and is believed to be due to the fact that hypercalcemia favors the deposit of calcium in the pancreatic ducts and the activation of pancreatic enzymes [12,13].

In addition, they may present neuropsychiatric manifestations such as depression, anxiety, fatigue, decreased quality of life, sleep disturbances and cognitive dysfunction, which are believed to be due to hypercalcemia [14].

Diagnosis

Because it can present asymptomatically or with nonspecific clinical manifestations, its diagnosis is laboratory-based. The first alteration that we will find is hypercalcemia, it is always prudent to confirm it with a second test where we will also include phosphorus, PTH (Parathyroid Hormone) and albumin to correct calcium in case of hypoalbuminemia; With this we will find: Elevated serum calcium, decreased phosphorus and elevated PTH. As complementary studies, kidney function must be measured, evaluating whether it has not been affected by hypercalcemia, blood count, alkaline phosphatase and urinary calcium excretion (increased by 25% to 35%), vitamin D levels (which can be elevated, and its deficiency). elevates PTH), bone mineral density (looking for osteopenia or osteoporosis), and renal imaging studies to evaluate stones or kidney damage [1,3,9].

It is important to differentiate from hypercalcemia associated with neoplasms, where calcium will be elevated and PTH decreased, and other alterations may also be found during the physical examination and laboratory and imaging studies. Once the diagnosis is confirmed with a laboratory, we can perform imaging studies, including neck ultrasound, tomography, magnetic resonance imaging, and technetium-99 scintigraphy; The objective is to look for parathyroid adenoma, to later decide the therapeutic approach. It should be clarified that imaging studies are not necessary to make the diagnosis [11,14].

Treatment

Treatment can be surgical or conservative, parathyroidectomy is the only definitive treatment, it is indicated in symptomatic patients, or in asymptomatic patients under 50 years of age with an increase in serum calcium greater than 1 mg/dl above its normal value, or with density bone mineral equal to or less than minus (-) 2.5 T score anywhere, and vertebral fractures; with creatinine clearance less than 60 ml/min, urinary calcium in 24 h greater than 400 mg/day, nephrolithiasis or nephrocalcinosis. With the procedure, the disease resolves, the symptoms subside, and calcium levels and PTH are normalized [14,15].

Conservative treatment can be performed in asymptomatic patients with high surgical risk, but periodic monitoring is important for the appearance of complications. The conservative option includes measures to avoid aggravating hypercalcemia as much as possible: Avoid thiazides, lithium, volume depletion, prolonged inactivity or bed rest, diet >1000 mg/day of calcium, maintain serum vitamin D3 levels of at least 20 ng/mL to 30 ng/mL, since its deficiency stimulates the production of PTH. Among the pharmacological options we have bisphosphonates, they are powerful inhibitors of bone resorption, so they can be useful to improve the BMD of HPP patients without surgery; and cinacalcet, thereby reducing serum PTH and tubular calcium reabsorption, thus reducing calcium. The follow-up of patients under conservative management consists of monitoring renal function for possible deterioration, annual or biannual evaluation of bone mineral density, periodic measurement of serum calcium and PTH [14,16].

Clinical Cases

Clinical case 1

77-year-old female patient, known to have hypertension, who came for medical control, treated with Olmesartan 40 mg, Nicardipine 10 mg, and hydrochlorothiazide 25 mg/day, without any cardiac or other symptoms. Laboratory studies are sent and show levels of vitamin D 10.6 ng/ml, serum creatinine 1.7 mg/dl, hemoglobin 12.5 g/dl. Vitamin D3 is started at 7,000 units per week and control studies are scheduled. He returned two months later with serum calcium of 11.9 mg/dl, albumin 4.5 g/dl, phosphorus 2.26 mg/dl (decreased) and vitamin D of 18 ng/ml, electrocardiogram was performed without alterations, ultrasound of the total abdomen with cholelithiasis and grade I fatty liver, without evidence of kidney damage, general urine without proteinuria. PTH levels are sent and monthly bisphosphonate is started, increased to 100,000 units of Vitamin D3 per month. PTH 163.30 pg/ml (15-65) was received by Electrochemiluminescence (ECLIA), calcium 10.78 mg/dl (after bisphosphonate) and creatinine 1.12 mg/dl (GFR by CKD-EPI 51 ml/min), neck ultrasound was indicated. There is no evidence of any mass or growth in the thyroid. Bone densitometry was indicated and the patient could not perform it due to costs. Conservative management was decided and an annual dose of zoledronic acid was indicated.

Clinical case 2

75-year-old female patient, who comes for her medical check-up, known for hypertension, COPD, obesity, and smoking, treated with perindopril, 10 mg, indapamide 2.5 mg, ibandronate 150 mg/month, vitamin D3 100,000 U/month, metformin 1000 mg/day, formoterol

4.5 mcg twice a day, and rosuvastatin 20 mg per day, with adherence to treatment. No signs of cardiac or pulmonary alteration, physical examination blood pressure 130/80 mmHg, weight 231 pounds with visceral obesity, rest without alterations. Tests with calcium 12 mg/dl (8.8-11 mg/dl), phosphorus 2.36 mg/dl (2.7-4.5 mg/dl), vitamin B12 294 mg/dl, and vitamin D 17.1 ng/ml suspected primary hyperparathyroidism.

In next evaluation with serum calcium 13 mg/dl, PTH 340 (15-65) pg/mL, phosphorus 2.38 mg/dl (decreased), glucose 104 mg/dl, Hba1c 6.2%, serum potassium 4.7 mg/dl, sodium 138 mg /dl, and creatinine 1.04 mg/dl. Electrocardiogram with sinus bradycardia, without QT alterations. Abdominal ultrasound with fatty liver, thyroid ultrasound with 1.4 cm × 1.6 cm nodule, referred for biopsy and a goitrogenic nodule, PA chest X-ray with grade I cardiomegaly. Conservative management was decided and an annual dose of zoledronic acid was indicated.

Discussion

Both cases are women, over 70 years of age, completely asymptomatic, and the finding was incidental when evaluating serum calcium levels, as reported in the literature; with multiple comorbidities, which is not unusual at that age, the first case with a slight elevation in creatinine, which subsequently improved, and without evidence of kidney damage in imaging studies. In PHPT one must always look for kidney damage due to nephrolithiasis and nephrocalcinosis [10].

Our cases presented elevated serum calcium, hypophosphatemia, and elevated PTH, which confirms the diagnosis. Both with low levels of vitamin D, and neck ultrasound without evidence of parathyroid adenomas. It is important to emphasize that the diagnosis is with laboratory studies, imaging studies are for surgical decision [5,14].

The management in both cases is conservative, they are older adults, with multiple comorbidities, which increases the surgical risk; They do not have symptoms of hypercalcemia and only one of the cases has altered renal function, but imaging studies showed no nephrolithiasis or nephrocalcinosis. There are also no pathological fractures. Despite suffering from primary HTN, in both cases the electrocardiogram did not show ventricular growth, arrhythmias or cardiac ischemia.

They are managed with bisphosphonate and vitamin D3 supplements in view of the deficiency they present. They remain vigilant with their controls in the outpatient clinic hoping there is no deterioration. It has already been reported in the literature that cases like these, in older adults with multiple comorbidities, management can be conservative [16].

Conclusion

screening must be done by measuring calcium in patients over 60 years of age. If it is elevated, it is prudent to corroborate and evaluate phosphorus and PTH. If there is elevated calcium and PTH with low phosphorus, we are diagnosed with PTSD. Imaging studies are used to look for parathyroid adenoma for surgical management if the case warrants it. Kidney, bone and cardiovascular damage must always be evaluated. In symptomatic patients, under 50 years of age, or with vertebral fractures, management is surgical. In older and asymptomatic patients, it is conservative, but monitoring the evolution.

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